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Foreign Animal Disease Report

United States Department of Agriculture
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Veterinary Services

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Current Events

Avian Influenza in Pennsylvania

Avian influenza virus H5N2 has been detected in eleven flocks of chickens and one with turkeys located in Snyder, Schuylkill, Fulton, and Northumberland Counties, Pennsylvania, one small broiler flock in Bristol County, Massachusetts, three flocks in Monmouth County, New Jersey, and a broiler flock on Long Island, New York. This is the same antigenic subtype of the virus that resulted in the destruction of more than 17 million chickens, turkeys, and other poultry in southeastern Pennsylvania, Virginia, Maryland, and New Jersey during 1983-84. However, laboratory tests of the virus isolated in January 1986 showed it to be not highly pathogenic. (See 12-2:5.) State agricultural officials quarantined the premises and destroyed the affected chickens and turkeys. State and Federal officials were continuing epidemiological tracing at the time this report was sent to the printer. Preliminary studies suggest that the movement of contaminated crates and trucks associated with marketing live poultry was the means for the introduction of the virus. (Adapted from APHIS Information Service news release.)

Emergency Disease Investigations, 1985

There were 480 diagnostic investigations for possible foreign animal diseases in the 50 States and Puerto Rico during fiscal year (FY) 1985 (October 1, 1984, through September 30, 1985). Of these, 440 cases were reported to the United States Department of Agriculture because of suspected vesicular conditions in cattle, horses, sheep, goats, and swine. Four hundred and thirteen of the investigations were made in the last quarter of the FY as a result of the recurrence of New Jersey type vesicular stomatitis (NJVS) in New Mexico, Arizona, and Colorado. (See 13-4.) Laboratory studies included the testing of serums from animals located in different areas to determine the geographic distribution of NJVS in Colorado and New Mexico. Possible insect vectors and affected livestock were also studied in cooperation with the National Centers for Disease Control (NCDC) and the

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Agricultural Research Service (ARS). Results of the studies were not available at the time this report was prepared.

A stallion imported from West Germany was found to be infected with contagious equine metritis (CEM) during May 1985 while being held in quarantine. A great deal of treatment effort was necessary to rid him of the disease. The quarantine was then released. The fractious nature of the animal and a localized secondary infection of the prepuce complicated the treatment.

In October 1984, a pet bird shipped from California to Hawaii was found to be infected with velogenic viscerotropic Newcastle disease (VVND). Surveillance tracing failed to detect further infection. Pet birds were also found to be infected with VVND on three Florida premises from January 17 to February 26, 1985. Two of the positive premises were commercial wholesale facilities. The third was a private aviary. Tracing of sales from the positive premises disclosed an additional case in Puerto Rico. Tracing of birds from the Puerto Rican premises and from the wholesale dealer in Florida to 25 additional States failed to disclose additional cases.

In May 1985, a newly purchased pet bird in a private home in North Carolina was found to be infected with VVND. The virus was also found at the bird's source in Missouri. (Dr. Arthur E. Hall, 301 436-8073.)

Puerto Rico Tick Program Update

Specialized training was given to the State and Federal personnel who recently conducted a tick surveillance effort in eastern Puerto Rico. Infested premises were identified and treatment started to eliminate ticks. Surveillance data will be computerized as a first step in the automation of all tick program data. A technical review is scheduled for March 1986.

Tick problems in Puerto Rico are complicated by bovine babesiosis (cattle tick fever). (See 14-4.) By January 23, 1986, a total of 314 premises with approximately 26,676 cattle were under quarantine for babesiosis or exposure to vector ticks. Fifty-four of the herds were diagnosed as infected with babesia based in part upon the results of laboratory examinations of stained blood smears.

Pesticide treatment to destroy ticks in infected herds is being done at 14-day intervals. (Dr. G. P. Combs, 301 436-8097.)

FMD Surveillance in Mexico

Surveillance activities of the Mexican-American Commission for the Prevention of Foot-and-Mouth Disease (CPA) during 1985 on a total of 119 reports of vesicular diseases in Mexico led to the diagnosis of New Jersey type vesicular stomatitis (NJVS) in 49 herds and Indiana type vesicular stomatitis (INVS) in four herds.

While VS was seen mainly in bovines, in three of the affected herds, horses also had VS. Horses were not affected on 25 premises with bovine VS. On 12 premises with bovine NJVS, where pigs were present, none of the pigs were affected by VS. In 1984, the investigation of 185 reported occurrences of vesicular diseases disclosed NJVS on 94 premises and INVS on one premises. (See 13-1.)

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The largest number of affected herds was found in enzootic areas of Veracruz and Guerrero. Foci of affected herds were seen in Tierra Blanca, Veracruz, and in Ometepec and Ayutla, Guerrero. For the first time, NJVS was found in Baja California Sur, in the Municipio of Mulege.

The average attack rate for NJVS in affected bovine herds for lesion cases was 8.0 percent, compared with 6.1 percent in 1984. The attack rate for INVS was 11.5 percent. Attack rates in individual herds ranged from 1 percent to 100 percent. Although one death reportedly was associated with NJVS, it would be difficult to confirm that this death was due to VS.

In only 3 of the total 49 herds investigated was there any introduction of cattle during the month before onset of NJVS. In the remaining 46 there was no history of the introduction of cattle to explain the occurrence of VS.

Most of the occurrences of NJVS were in herds in which the disease had not been seen previously. Owners of 7 of 37 herds with NJVS reported having similar cases before 1985. Also, information obtained during owner interviews suggested that there were large numbers of unreported cases in the vicinity of herds known to be infected.

Of the total 50 affected bovine herds, VS lesions were reported in the mouth in 70 percent, in both mouth and on teats in 20 percent, on teats only in 6 percent, and in both the mouth and on feet in 4 percent. Lesions on the feet only were not reported. (Adapted from the 1985 report of the Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease.)

AI Seminar in Mexico

The Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease (CPA) and the United Nations Food and Agriculture Organization (FAO) conducted a very successful seminar and test exercise for avian influenza (AI) November 23-30, 1985, in Tehuacan, Mexico. Fifty-nine animal disease specialists from Mexico and 16 countries in Central and South America participated. Objectives of the event were to improve the Mexican animal disease emergency response system, evaluate the country's emergency plan for AI, use the training technology developed by CPA, transfer up-to-date information on AI, and exchange national emergency plans. Two days were spent in discussing AI and means to control and eradicate it if it does enter a free area such as Mexico. The remaining 3 days were dedicated to the simulation of an AI outbreak in an imaginary county, to give the participants the experience of dealing with such an emergency. (Adapted from reports received from Dr. John Mason, Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease, 905 531-7600.)

FAD Training Program

November 1985 marked the first foreign animal disease course held at Plum Island for teachers of foreign animal disease courses at colleges of veterinary medicine. Eighteen professors, representing 16 veterinary schools, participated.

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Two seminars were planned for FAD diagnosticians. The first was in Lincoln, Nebraska, during the first week in February 1986. It was attended by approximately 55 diagnosticians from the VS Western and Central regions. The second seminar will include approximately 50 diagnosticians from the VS Northern and Southeastern regions and International Operations. The second seminar will be held during May in Hyattsville, Maryland.

Two foreign animal disease schools are planned. The first will be during April, the second in September.

A wildlife disease seminar for FAD diagnosticians is scheduled at the University of Georgia during late May. (Dr. Wesley H. Garnett, 301 436-8091.)

World Animal Disease Roundup

The year 1985 ended with few new developments as far as internationally important animal diseases are concerned. The **foot-and-mouth disease** (FMD) situation in Italy suffered a setback with the occurrence of virus Type C in swine during November. All previous cases in the Italian FMD epidemic were caused by virus Type A. No other cases of FMD were reported from Europe in 1985. However, Europeans keep a close watch on Turkey, troubled by outbreaks caused by viruses Type O_1 and A_{22}. There is also apprehension because of the threat of Type $Asia_1$, reported lately from Iraq and Malaysia. Elsewhere in Asia, FMD was reported from Hong Kong, Thailand, the Philippines, Oman, and Israel. In the Americas, FMD was reported from Argentina, Brazil, Colombia, Ecuador, Peru, and Venezuela. Numbers of cases in South America are presently decreasing. Chile has reported no FMD for over 18 months.

Swine vesicular disease (SVD) has not been reported since a case in Germany in October 1985. (See 13-4.) However, recent reports indicate the Commonwealth Agricultural Bureaux Laboratory at Pirbright, England, isolated SVD virus from material submitted from Hong Kong in August 1985. **African swine fever** and **hog cholera** (HC) remained relatively static toward the end of 1985, but there was a slight decrease in the number of HC cases reported. Whether this is a beginning payoff for European eradication efforts remains to be seen.

Also of interest are reports on some conditions not heard from for awhile: A case of **Rift Valley fever** was reported in South Africa, a case of **African horse sickness** was reported in Botswana and one in Zimbabwe, and several cases of **lumpy skin disease** were reported in Kenya. (Dr. Hans J. Seyffert, 301 436-8285.)

Focus on... Maedi and Related Diseases of Sheep

Maedi initially was described as a slow progressive interstitial pneumonitis of sheep in Iceland and at that time was thought to be a separate distinct entity (G. Gislason, 1947). A chronic encephalitis with severe wasting was described later in Icelandic sheep and called visna (B. Sigurdsson, P. A. Palsson, and H. Grimsson, 1957).

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The two are now known to be different manifestations of a common viral infection and are referred to as maedi-visna. This name is used throughout most of the world for similar diseases.

Although maedi-visna has not been reported in the United States, a similar disease has been called progressive pneumonia (H. Marsh, 1923 and 1966), with various modifiers such as Marsh's Montana, chronic, and ovine (E. V. Cowdry and H. Marsh, 1927; C. T. Creech and W. S. Gochemour, 1936; R. C. Cutlip, T. A. Jackson, and H. D. Lehmkuhl 1979). In this review, we use ovine progressive pneumonia (OPP) to refer to this disease and briefly discuss the history, cause, transmission, susceptible species, clinical signs, lesions, pathogenesis, diagnosis, serology, prevalence, treatment, and control of maedi-visna, OPP, and their causal viruses.

Maedi-visna and OPP are caused by closely related retroviruses that induce severe and progressive emaciation, dyspnea, paralysis, lameness, and indurated udders related to lymphoproliferative and degenerative processes. Sheep that are infected with either virus are carriers for life, even in the presence of high levels of circulating antibodies. Most animals never show evidence of disease. Even though the diseases are similar in most respects, the etiologic viruses differ slightly in nucleic acid sequence. The significance of this difference is unknown, but may represent only strain and not species differences. The two diseases can be discussed together because of the close similarity of the causal agents, clinical signs, and lesions.

History

The respiratory form of OPP was first described in the United States in 1923 in sheep from Montana and was called progressive pneumonia. A report from South Africa in 1915 described a similar disease, later called Graaff-Rinet disease after the region where it was found (D. T. Mitchell, 1915). The diseases that were described in the United States and South Africa were compared and considered identical (E. V. Cowdry and H. Marsh, 1927). Apparently, some sheep had both OPP and pulmonary adenomatosis (jaagsiekte) that led to lingering confusion about the two diseases. We now know that they are distinct entities: **Maedi-visna and OPP are lymphoproliferative and degenerative diseases, and adenomatosis is a carcinomatous disease.**

Maedi-visna was described in France in 1940 and called bouhite, a local name for pulmonary lymphoma, and in Holland in 1943 as zwoegerziekte, meaning lagging sickness (F. Lucam, 1942; H. Koens, 1943). In Iceland, the pulmonary form of the disease was described in 1947 under the name maedi, meaning dyspnea, and the paralytic form of the disease was described in 1957 under the name visna, meaning wasting. The diseases were eradicated from Iceland by an extensive slaughter program. They were later shown to be caused by the same virus.

Causes

Maedi-visna and OPP are caused by nononcogenic, exogenous retroviruses of the subfamily Lentivirniae (F. Fenner, 1976). The viruses are closely related in morphological, chemical and serological properties; (L. B. Stone, K. K. Takemoto, and

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M. A. Martin, 1971; M. J. Weiss et al., 1977); however, minor differences in nucleic acid sequence have been shown with nucleic acid hybridization techniques (M. J. Weiss et al., 1976; S. M. Roberson, 1982). There is also morphologic similarity and some nucleic acid homology with the viruses of caprine arthritis-encephalitis (CAE) of goats (J. E. Dahlberg, J. E. Gaskin, and K. Perk, 1981) and acquired immune deficiency syndrome (AIDS) of man. (I. M. Chiu et al., 1985; M. A. Gonda et al., 1985; P. Sonigo, 1985). The CAE virus also has close antigenic similarity with maedi-visna and OPP (J. E. Dahlberg, J. M. Gaskin, and K. Perk, 1981). However, humans are not known to be susceptible to the viruses of CAE, maedi-visna, or OPP.

Even though there is morphologic similarity, there is no antigenic relationship with the oncogenic retroviruses (D. H. Harter et al., 1973; L. Stowring, A. T. Haase, and H. P. Charman, 1979). Maedi-visna and OPP viruses consistently cause syncytial formation in cell culture (B. Sigurdsson, P. A. Palsson, and H. Grimsson, 1957), but vary in their cytolytic properties from strain to strain (personal observation). Permissive replication of the viruses can occur in many cell types from different species (D. H. Harter, K. C. Hsu, and H. M. Rose, 1968; C. Torchio, and R. S. Trowbridge, 1977; R. S. Trowbridge, J. F. Schneider, and R. Haddad, 1982). Choroid plexus and fetal lung cells from sheep are most commonly used for virus replication. Virus can either become latent or persistent in cells in culture and in the animal host (V. Barban et al., 1984; A. T. Haase, and H. E. Varmus, 1973). This occurs because the virus forms a provirus; i.e., integration of viral genetic material into that of the host cell. The provirus can lie dormant for an undetermined length of time, probably for life, even in the presence of circulating antibodies. Thus, seropositive sheep are virus carriers.

Transmission

Virus is transmitted to susceptible sheep primarily through the colostrum (G. F. DeBoer, 1970; P. A. Palsson, 1976). Mononuclear cells carrying the provirus in colostrum of infected ewes are probably passed through the intestinal epithelium of the neonate to establish infection. Contact transmission does occur when animals are closely housed, but is less important than milk transmission (B. Sigurdsson, P. A. Palsson, and A. Tryggvadottir, 1953). Transmission in utero can occur, but is rare (R. C. Cutlip, H. D. Lehmkuhl, and T. A. Jackson, 1981).

Susceptible Species

Sheep and goats are the only species known to be susceptible to OPP and maedi-visna (K. L. Banks et al., 1983). Attempts have been made to infect a number of laboratory and other domestic animals, but all have failed (P. A. Palsson, 1976; R. C. Cutlip and H. D. Lehmkuhl, in preparation). There is recent evidence of a distinct breed difference in susceptibility to development of disease, but probably not to infection (R. C. Cutlip, H. D. Lehmkuhl, and K. A. Brogden, In Press). Sheep of the Border Leicester breed were shown to be significantly more susceptible to disease than sheep of the Columbia breed. Circumstantial evidence is accumulating that some other breeds of sheep are highly susceptible to development of disease.

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Clinical Signs

Signs develop and progress slowly in both diseases until death ensues after several months to a year (R. C. Cutlip, T. A. Jackson, and H. D. Lehmkuhl, 1979; G. F. DeBoer, 1970; P. A. Paulssen, 1976; R. E. Oliver et al., 1981). Progressive emaciation, in spite of a normal appetite, is frequently the earliest sign. Dyspnea, especially during exercise, is a common finding. Lameness with swollen joints, especially the carpal and tarsal joints, and posterior weakness with ascending paralysis is seen less frequently (R. C. Cutlip et al., 1985a). Lameness and weakness are progressive and may last for months to years. A hard, indurated udder with reduced milk production is sometimes associated with infection (R. C. Cutlip et al., 1985b). Most fatally infected sheep die from secondary bacterial pneumonia. We emphasize that most sheep which are infected with the viruses of maedi-visna or OPP do not succumb to the clinical disease, but once signs are seen there is a relentless course to death.

Lesions

The basic lesion in all affected tissues is the accumulation of lymphatic tissue that often forms nodules that may contain germinal centers. Lungs are enlarged and firm with multiple grey areas and weigh three to four times normal. Lymphatic tissue accumulates in the pulmonary interstitium around airways and blood and lymphatic vessels. In some cases the alveolar epithelium becomes hyperplastic and there is excess fibromuscular tissue. Lesions within the central nervous system consist of varying degrees of accumulation of lymphocytes in the meninges, choroid plexus, and white matter of the brain and spinal cord. In severe cases, they are accompanied with primary demyelination and necrosis. The subependymal and subpial areas of the brain are most involved. Arthritis commonly affects the appendicular joints, but may involve other joints. The lesions are extensive proliferation of synovial and bursal membranes, fibrosis of the joint capsule, and degeneration of articular cartilage and bone. Affected joints are dry (nonsuppurative) and contain some fibrin. The proliferated synovial membranes are hyperemic and infiltrated with lymphocytes, plasmacytes, and macrophages. Lymphocytic mastitis, with multiple accumulations of lymphocytes and plasmacytes in the interstitium and breaks in ductal and acinar epithelium, is a common feature. Fibrinoid necrosis in the tunica media and mononuclear cell accumlations in the tunica adventitia and tunica media are occasionly seen in small muscular arteries of joint capsules, kidneys, brain, and other tissues.

Pathogenesis

Pathogenesis of maedi-visna and OP is best explained as a result of chronic antigenic stimulation related to persistent viral infection. Immunization of infected sheep with visna virus was shown to hasten the development of lesions and, conversely, immunosuppression of infected sheep was shown to retard the development of lesions (N. Nathanson et al., 1981 and 1976). Persistence of the viruses is related to integration of viral nucleic acid into host cell nucleic acid (provirus formation) (A. T. Haase and H. E. Varmus, 1973). The provirus has recently been shown to reside in mononuclear macrophage-type cells of the lungs and in glial cells in the brain (O. Narayan et al., 1982; L. Stowring et al., 1985). Antigenic drift of the virus may also contribute to the persistence, but is less important than integration of viral nucleic acid into host cell nucleic acid (M.

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Gutnadottir, 1974; O. Narayan, D. E. Griffin, and J. Chase, 1977; R. Lutley et al., 1983).

Diagnosis

Because of the persistence of virus in the presence of circulating antibodies, a diagnosis of infection by maedi-visna or OPP virus can be made by identifying either virus or specific antibody in the blood. A tentative diagnosis of disease is made from clinical signs and either identification of serum antibodies or isolation of virus. Definitive diagnosis is dependent upon finding characteristic lesions at necropsy in conjunction with clinical signs and demonstration of virus by either serology or isolation.

Serology

Currently, three serologic tests are used to detect antibodies to maedi-visna and OPP viruses: An agar gel immunodiffusion test (AGIDT), enzyme linked immunosorbency assay (ELISA), and indirect immunofluorescent test (IIFT) (R. C. Cutlip, T. A. Jackson, and G. A. Laird, 1977; D. J. Houwers, and A. L. J. Gielkens, 1979; P. Bront, G. Charlier, and A. DeSmet, 1981). The three tests vary only slightly in sensitivity and specificity, but the simplicity of the AGIDT makes it the test of choice for most surveillance or control programs (M. Dawson, 1982). Actively acquired precipitating antibodies to maedi-visna and OPP viruses are slow to develop and with one known exception are maintained for the life of the animal (G. F. DeBoer, 1970; R. Lutley et al., 1983; L. Sihvonen, T. Estola, and J. Tuomi, 1980). Thus, actively acquired antibodies indicate that the animal is infected with virus. Passively acquired antibodies are obtained through the colostrum and are lost by 6 months of age. Therefore, serologic tests are uninterpretable during the first 6 months of life. During this time, a positive test indicates that the animal is either infected and has produced antibodies or has passive antibodies; a negative test indicates that the animal is either noninfected or is infected and has not yet produced antibodies. After 6 months of age, a positive test indicates that the animal is persistently infected; a negative test is interpreted the same as one before 6 months of age. Some seropositive sheep or goats will become seronegative following parturition because of the large quantity of antibodies lost in the colostrum. Because of these possibilities, serologic testing is best done on a flock or herd basis.

Prevalence

Prevalence of infection and of disease vary greatly. Retroviruses of sheep are reported worldwide except in Iceland, Australia, and New Zealand. Ovine progressive preumonia has been reported in the United States and Israel (R. C. Cutlip, T. A. Jackson, and G. A. Laird, 1977; N. L. Gates et al., 1978; E. M. Huffman et al., 1978; E. M. Huffman et al.,). In the United States, as many as 50 percent of the total sheep population may be infected. (R. C. Cutlip, T. A. Jackson, and G. A. Laird, 1977; N. L. Gates et al., 1978; E. M. Huffman et al., 1981). Within individual flocks, infection increases with age. This may indicate lateral transmission of virus or delayed seroconversion after infection at birth. The percent of infected sheep that develop clinical disease is not known, but as stated, certain breeds of sheep are more susceptible to disease than others (R. C. Cutlip, H. D. Lehmkuhl, and K. A. Brogden, in press).

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Treatment

No effective treatment for the viral infection is known, so treatment must be symptomatic. Anti-inflammatory drugs can be used to reduce joint pain and antibiotics can be used to prevent secondary bacterial infections, especially of the lungs.

Control

Vaccines against maedi-visna and OPP have not been successful (R. C. Cutlip, et al., in preparation), but several laboratories, including the Agricultural Research Service laboratory at Ames, Iowa, have eradicated maedi-visna and OPP viruses from experimental sheep by collecting offspring at birth and rearing them in isolation either on pasteurized milk or a milk substitute (M. R. Light et al., 1979; D. J. Houwers et al., 1983). We and others have also eradicated the viruses from experimental sheep by a test and removal procedure (D. J. Houwers, J. Schaake, Jr., and G. F. DeBoer, 1984; R. C. Cutlip and H. D. Lehmkuhl, In Press). We recommend the following two methods of control:

Method 1. Test and remove.

1. Bleed all sheep and goats in the flock and test serologically for antibodies to maedi-visna and OPP viruses. All sheep and goats on the farm must be included because both viruses can infect either species and cause a positive reading of the serological test.

2. Remove from the flock all seropositive animals and their progeny that are less than 1 year old. Animals removed from the herd can either be sold for slaughter or isolated in separate facilities. Slaughter is recommended because of the danger of cross contamination.

3. Keep the clean flock isolated from infected sheep and goats and from people and equipment in contact with an infected flock.

4. Add only seronegative animals to the flock, either from other seronegative herds or from seronegative parents in an infected flock following 1 year of isolation with a negative test reading.

5. Testing must be done annually until there are at least two consecutive negative flock tests to be reasonably sure that the flock is free of the virus.

Method II. Isolate and artificially rear progeny.

1. Remove progeny from dams before nursing and maintain in isolation.

2. Proceed with steps 3 and 4 described in test and removal method above to assure virus free status of the new herd. Annual serologic tests are recommended.

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General References on Maedi-Visna and OPP

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(Randall C. Cutlip, H. D. Lehmkuhl, and M. J. F. Schmerr, USDA Agricultural Research Service, Ames, Iowa 50010; 515 239-8200.)

Questions about the FAD Report may be sent to:

Dr. E. I. Pilchard
APHIS, USDA, VS
Room 760, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

Thirty days before moving, send address change, and if possible, mailing label from latest issue to:

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6505 Belcrest Road
Hyattsville, MD 20782

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Current Events

Dogs are being used in the passenger arrival areas of international airports to find prohibited fruit and meat in baggage arriving from foreign countries. The detector dog program was launched July 12, 1984, at four airports: Los Angeles International, San Francisco International, Houston Intercontinental, and John F. Kennedy International. Beagle dogs wearing bright green U.S. Department of Agriculture jackets do the sniffing. Kept on leashes and under the constant supervision of their handlers, they have been trained to

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immediately sit when they smell citrus fruit, mangoes, beef, sausage, or other agricultural products. Their handlers then reward them with a small treat. A quarantine tag is placed on each suspicious bag or parcel and a green "A" is marked on the Traveler's Customs Declaration Card. Primary inspection by a U.S. Customs Service inspector is followed by an evaluation of the accuracy of the alert, by a Plant Protection and Quarantine (PPQ) officer.

Six additional detector dog teams are scheduled for 3 months of training at the U.S. Air Force Dog Training Center at Lackland Air Force Base in San Antonio, Texas. Following training, they will go into action at major ports of entry, such as Chicago, Illinois, and Miami, Florida.

Besides being extremely cooperative, beagle hounds have an extraordinary sense of smell. They are gentle with humans and have a puppy-like nature. They don't become nervous or excited when surrounded by the kind of commotion found in baggage inspection areas.

The dogs usually rest for 20 minutes during each hour, or for longer periods if they show signs of tiring or lose interest in their work. They stay with their handlers throughout the work day and are kept in kennels overnight. The dogs' expected working life is from 8 to 10 years. The handler who has been with each dog last has the option of keeping it as a pet.

Two of the detector dogs whose effectiveness was recently evaluated had a success rate approaching 70 percent on baggage from European flights, meaning that 70 percent of their "alerts" were on baggage containing prohibited material.

Civil penalties for amounts up to $50 are imposed on travelers who fail to declare or attempt to smuggle prohibited agricultural products.

In addition to detector dog teams, sophisticated x-ray machines are being installed at certain high-risk airports to enhance the U.S. Department of Agriculture's ability to intercept nondeclared, prohibited plant and animal products. (Dr. Robert R. Ormiston, 301 436-8065)

World Animal Disease Roundup

African swine fever (ASF), appeared for the first time in the Netherlands during March 1986. Feeding of uncooked garbage containing ASF virus is thought to be the means of its introduction. Another notorious foreign disease, **hog cholera** (HC), reappeared in Great Britain, which had been considered free of the disease since 1971. Otherwise, the incidence of HC worldwide appears to be declining. **Swine vesicular disease** has not been reported anywhere since October 1985, when it was diagnosed in West Germany.

The incidence of **foot-and-mouth disease** (FMD) remained static over the last 4 months. The disease surprisingly did not spill over from Italy into the rest of Europe. The epizootic caused by FMD virus type C seems to be declining after the vaccination of

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about 780,000 swine using monovalent type C vaccine.

Some other diseases not heard from for some time are being reported again. There were several occurrences of **Rift Valley fever** in South Africa and **African horse sickness** was diagnosed again in South Africa and Namibia. **Lumpy skin disease** was reported from Zaire, Madagascar, and South Africa. Norway reported **contagious equine metritis** for the first time in January 1986. (Dr. Hans J. Seyffert, Veterinary Attache, U.S. Embassy, Manila, The Philippines)

Imported Hides and Trophies

Veterinary Services (VS) is concerned with the possible introduction of exotic animal diseases into the U.S. through the importation of animal hides and trophies. The importation of hides of ruminants and swine, and trophies of wild animals from FMD countries is restricted by Federal regulations. These restrictions, in effect for many years, require the completion of specified procedures to destroy foreign pathogens.

Hides can be imported without restriction if:

1) the hides originate from and are shipped through countries free of FMD and rinderpest, or

2) they are hard dried ruminant hides, or

3) the hides are from ruminants and are packed wet in solutions of salt and mineral acid in leak-proof barrels, casks or cases, or

4) the hides are from ruminants and have been dehaired with lime for immediate manufacturing into rawhide.

If hides destined for the United States have not met the above requirements, they may be permitted to be taken directly from the port of arrival to a Veterinary Services (VS) approved establishment for further processing.

Wet or fresh ruminant hides must be soaked in a solution of sodium biflouride for at least 24 hours. Dry swine hides must be soaked in either a heated water bath, or a molar salt solution. However, if the swine hides are wet they must be soaked in a solution of sodium bifluoride.

Animal trophies that are fully tanned and ready for display, including horns, antlers, and clean dry bones for museum purposes, may be imported without restriction.

Bones, hoofs, antlers, and horns that are not dry and free of flesh and sinew, or that are used in industrial processes that require cutting and grinding (knife handles, belt buckles, etc.), must be boiled or heated with steam for 30 minutes.

If an importer cannot or will not comply with the above requirements then the hides or trophies are refused entry at the port of arrival. The importer has the option of returning the hides or trophies to the country of origin or having them

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destroyed. The importer must pay the costs of return or destruction.

Regulations are being amended to prevent the introduction of exotic ticks with imported trophies and hides. These amendments and other changes in regulations are needed from time to time to ensure that import requirements reflect the current status of knowledge on exotic disease prevention. (Dr. Michael J. Gilsdorf, 301 436-8383)

International Reference Laboratories

The appearance of animal diseases in susceptible populations can be devastating to agricultural societies. They may have serious consequences not only to the owners, who are immediately concerned, but also to urban dwellers through the effects of resulting shortages. They affect national economies through interference in international commerce.

By actively assisting governments to respond and recover from the effects of severe animal disease outbreaks, the Food and Agriculture Organization of the United Nations (FAO) has supported the development of disease diagnosis, vaccine production, and strategies to deter the effects of diseases, particularly the economically more devastating ones.

In a recent effort to intercept the movement of animal diseases, FAO has identified specific laboratories with recognized capability for dealing with certain diseases, to serve as Regional Reference Centers and provide training and service, in addition to their normal national responsibilities. The objective is to identify centers of excellence that are technically able and politically willing to assist other governments of the region to deal with invasion by a disease that provides a new and unique challenge to their resources.

For example, the United States Department of Agriculture (USDA) Foreign Animal Disease Diagnostic Laboratory (FADDL) at Plum Island, New York, has been designated as the FAO Reference Center for the world for African Swine Fever; for the Western Hemisphere, for several diseases considered exotic to the region; and for Central and North America, for vesicular diseases, including FMD. The USDA National Veterinary Services Laboratories at Ames, Iowa, has accepted the diagnostic responsibility for the Western Hemisphere for Newcastle disease and avian influenza and for North America for hog cholera.

To assist FAO in carrying out the review of potential reference centers, William M. Moulton, Clinical Professor, International Veterinary Medicine, Tufts University School of Veterinary Medicine, recently visited 26 laboratories in 20 countries throughout Asia, Australia, Eastern and Western Europe, Latin America, and North America to evaluate their technical capability, resources, bio-containment, and to determine the appropriate region to be serviced. (Dr. Moulton may be contacted at P.O. Box 36, East Dover, VT 05341, 802 348-7996)

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Mexican Border Security

Surveillance activities by APHIS, VS help assure that foreign animal diseases and pests will not cross the Texas-Mexico border. The resultant security is actually the result of a concerted effort involving more than one unit of VS. A Tick Eradication Program sends mounted inspectors along the banks of the Rio Grande River each day to prevent stray animals from entering the United States. In the course of these patrols, the health of local livestock is observed and any significant abnormality noted for possible followup action by a team from the Border Surveillance Unit.

The surveillance team is comprised of six animal health technicians, a secretary, and a veterinarian. Each technician is assigned a district as his primary responsibility. He maintains liaison with the county extension agent, agricultural teachers, veterinarians, feed store operators, farmers, and ranchers. Any unusual event associated with livestock health is investigated.

If warranted, the premises of concern may be visited. The surveillance extends from Brownsville to Laredo, Texas.

Sera are collected from all Rio Grande Valley swine as they are slaughtered. Most of the marketed swine are slaughtered in San Antonio on Monday of each week. Veterinary Services technicians collect and identify the sera, then submit them to the National Veterinary Services Laboratories (NVSL) at Ames, Iowa, where they are tested for hog cholera and African swine fever antibodies. In the event of a positive test result, a visit is made to the premises of origin to examine the herd and determine possible followup actions.

Veterinary Services personnel regularly inspect all swine garbage feeding operations near the Mexican border. All swine and poultry producers are contacted routinely and the health status of their livestock is evaluated.

Prompt action by surveillance teams has prevented the spread of hog cholera and exotic Newcastle disease from local outbreaks.

The cooperative effort that exists between the Tick Eradication Program, Texas Animal Health Commission, Border Surveillance Unit, and the livestock industry assures an effective surveillance program. (Dr. Victor H. Driscoll, USDA, APHIS, VS, Laredo, TX)

Swine Exported to China

The fourth and by far the largest shipment of breeding swine from the United States to the People's Republic of China has been released from quarantine, continuing the development of trade between the two nations.

The shipment of 1,836 swine from Kentucky arrived in Guangzhou, China, in December and was immediately placed in post-entry quarantine for observation and testing.

The Chinese are interested in importing animals from the United States to introduce new breeding stock and to increase their

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production. They are especially interested in swine because of the high quality of U.S. pork and pork products.

Under a 1984 agreement, the Chinese require imported U.S. livestock to undergo rigid quarantine, testing, and shipping procedures.

The swine just released from quarantine were tested for several diseases on the farms of origin in the U.S. before being isolated from other animals for 30 days while they were retested prior to shipment. The swine were then flown to China where they were placed in quarantine for 45 days during which they were tested again to assure the Chinese that the animals were healthy before release.

A November 1985 meeting in China between USDA and Chinese representatives led to the renegotiation of some of the testing requirements that will simplify the process of exporting swine in the future. Preliminary discussions also were held on importing Chinese swine into the United States and exporting bovine semen and embryos from the United States to China.

Chinese swine produce large litters and are highly desirable for cross breeding. No agreement has yet been reached on exportation of U.S. bovine semen and embryos to China, but discussions will continue.

The 1984 agreement between China and the United States opened the door for trade between the two nations in cattle, poultry, and swine.

There is intense interest in U.S.-Chinese trade in livestock. China has a great potential as an export market for animals from the United States. Over 1,190 cattle were shipped on seven flights throughout 1985. Two more shipments of Holsteins, 173 from Wisconsin and 180 from Pennsylvania, were exported in January 1986. (Adapted from news release from USDA News Division, 301 436-7799)

Avian Influenza Virus Genetics

A recently reported investigation studied the genetic origin and genetic changes that led to the appearance of the highly virulent influenza virus strain H5N2 of chickens (Chicken/Pennsylvania/1370/83). (Bean WJ, Kawaoka JM, Wood JM, Pearson JE, and Webster RG. 1985. Characterization of virulent and avirulent A/Chicken/Pennsylvania/83 influenza viruses: Potential role of defective interfering RNAs in nature. Virology 54:151-160.) The results of these studies showed that the virulent virus isolates were derived directly from preceding avirulent viruses. The avirulent virus, which first appeared 5 months before the detection of the virulent strain, was shown to be closely related to viruses commonly found in wild ducks and domestic turkeys.

The most probable sequence of events that led to its appearance is that a recombinant virus of the H5N2 serotype was generated in waterfowl and had limited ability to replicate in chickens. This virus was either introduced directly into a chicken population or alternatively, introduced into turkeys and then into the chicken

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population. The virus then replicated undetected for an unknown period of time--at least several months, possibly much longer. In April 1983, the virus had become well enough adapted to the new host to spread readily and cause recognizable clinical signs of disease. At this time the "avirulent" virus was isolated. Random mutations in the avirulent virus then generated the virulent virus. One critical mutation in the virus hemagglutinin has been located. The importance of other mutations in hemagglutinin and in the other genes is not known.

Although not directly related to the goals of the reported project, work with this virus has led to the development of a model system for the study of drug-resistant influenza virus strains, and it has provided a valuable model for the study of the genetic factors affecting virus virulence.
(William J. Bean, Jr., St. Jude Children's Research Hospital, 332 N. Lauderdale, P.O. Box 318, Memphis, TN 38101)

Focus on... African Swine Fever

African swine fever (ASF) is a contagious febrile, systemic, viral infection of swine caused by an Iridovirus. In the family Iridoviridae, ASF is the only virus that infects mammals; the others infect arthropods, amphibians, and fish. Until 1958, ASF occurred in Africa; then it was diagnosed in Portugal and was eradicated. The disease reappeared in Portugal in 1960, spread to Spain, and since then has been endemic in the Iberian Peninsula. Since 1960, ASF has occurred in several Mediterranean countries, France, and most recently (1985) in Belgium. Today in Europe, ASF is present in the Iberian Peninsula and Sardinia. In the Western Hemisphere, ASF first occurred in Cuba in 1971; it was eradicated. The disease was next diagnosed in Brazil in 1978, Dominican Republic in 1978, Haiti in 1978, and Cuba in 1979. Although these countries have subsequently announced that they have eradicated ASF, the United States has recognized only the Dominican Republic as ASF-free.

Reservoir - Epidemiology

For many years the reservoir for ASF in southern Africa was thought to be indigenous wild pigs, particularly the warthog, because they are susceptible but do not die as a result of ASF virus infection. The soft tick was identified as a vector. Then it was learned that once a tick was infected it would remain infected through the remaining stages of development (transstadial transmission), ASF virus would pass through the egg to the next generation of ticks (transovarial transmission), and female ticks could be infected by insemination by infected male ticks. When soft ticks from southern Africa are fed on an infected pig, a few ticks subsequently die; however, when soft ticks from other than southern Africa, e.g. southern California, are fed on an infected pig, a large percentage may die.

The role of the warthog now appears to be one of an amplifier of the virus. Baby warthogs in the burrow are bitten by infected ticks, develop a viremia, and are then a source of infection for noninfected ticks. It is believed that a virus concentration of

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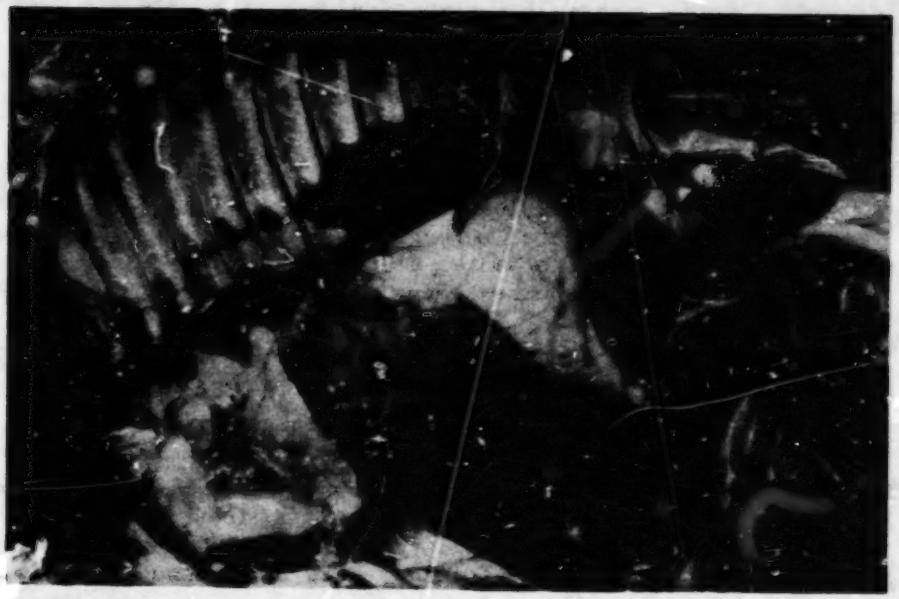
at least 100,000 infective units per ml blood is needed to infect arthropods. The adult warthog that has an antibody titer usually does not have a sufficiently concentrated viremia to infect ticks. In light of these facts, the consensus now is that ASF virus is a tick virus and the pig is an accidental host. Once a population of ticks becomes infected, it may remain infected for many years.

Disease Cycles

Since 1963, the incidence of ASF in the Iberian Peninsula has peaked about every 7 years. If the immunology of ASF were similar to other viral diseases, this cyclic incidence could be expected because ASF antibodies would cause a decreased incidence of disease and thus permit the development of a susceptible population. It is interesting to note that hog cholera has cycled about every 8 years in Europe. Although ASF antibody (IgG) does not neutralize ASF virus, it has been shown experimentally that the severity of the disease is significantly reduced in piglets that are born to and nurse a convalescent dam. Therefore, antibody does influence the course of the disease.

Clinical Forms of Disease

The clinical form of ASF in the Iberian Peninsula in 1958 has changed over the years. The disease that was introduced was highly virulent; this form of the disease is characterized by an almost 100 percent mortality 7-10 days after inoculation and is the form most frequently described in the literature. Today, ASF viruses isolated from the Iberian Peninsula can be classified as moderately virulent--the pigs will develop a high fever for 10-14 days or more but many will survive and mature, or as low virulent--the pigs may only seroconvert. The survival of infected pigs has resulted in the spread of ASF to other European countries and the Western Hemisphere. Recovered pigs are persistently infected and the virus will survive for some time in many products produced from these pigs. The spread of ASF to other countries usually has been due to the feeding of garbage containing ASF-infected pork.



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Classical Form

The classical clinical signs and lesions of highly virulent ASF are: high fever, reddened areas of skin, very enlarged dark friable spleen, very enlarged dark blood clot-like gastrohepatic lymph nodes, enlarged dark renal lymph nodes, petechiated kidneys, and edema of the lungs. Other lesions include hemorrhage on the peritoneum, heart, and renal pelvis, and edema of the gall bladder. Peripheral lymph nodes may be edematous. Mortality is usually 100 percent, 5 to 8 days after onset of fever. Little or no antibody is formed and tissues are positive by the direct immunofluorescent test up to the time of death.

Moderate Virulence

Clinical signs and lesions in moderately virulent ASF infection can be similar to but not as severe as those seen in the highly virulent form; the spleen may not be as large and retains normal color and texture, and the gastrohepatic and renal lymph nodes may not be as hemorrhagic. Mortality may be low. When death occurs, it is usually 9 days or more after the onset of fever.

In a breeding herd, abortion may be one of the first signs. At 6 days after infection, circulating antibody can be present and the tissues may be negative for ASF antigen by direct immunofluorescent test.

Some reasons for the differences in the above forms of the disease are: The highly virulent virus causes extensive necrosis, particularly in the spleen, and necrosis allows the spleen to become distended with blood; the moderately virulent virus infects the same cells but most survive; the difference in antibody production may result from the fact that the virus replicates preferentially in antigen-presenting cells of the mononuclear phagocytic system; the highly virulent virus kills these cells before the immune response is initiated, while cells infected with the moderately virulent virus retain some normal function and thus initiate antibody production. The immune response in pigs infected with a moderately virulent ASF virus is similar to that in other viral infections; IgM is present at 4 days and IgG at 6 days after infection. The peculiarity of ASF is that the circulating antibody does not neutralize the virus or eliminate the viremia.

Low Virulence

Low virulence ASF infection in growing pigs may cause only seroconversion. In a breeding herd, there may be abortions. To confirm ASF as the cause of abortion, at least a heparinized blood sample from the dam must be submitted to the laboratory in addition to fetal tissue because aborted fetuses are a poor source of virus.

* U.S. Government Printing Office : 1986 -490-918/40349

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Chronic

In chronic ASF, any or all of the following clinical signs and lesions can occur: fever, skin thickening and necrosis, arthritis, consolidated pulmonary lobules and necrosis, lymphadenopathy, and pericarditis.

Virus Replication

African swine fever virus replicates in the cells of the monomuclear phagocytic system. The virus does not replicate in epithelial tissue; therefore, the amount of virus exteriorized is low unless there is bleeding. Blood during the febrile period can have a viral titer of 100,000 infective units per ml or higher. It has been estimated that 90 percent of the virus in the blood is attached to the erythrocytes.

One surprising feature in an ASF infection is that pigs can have a fever of 41°C (106°F) or higher for a week or more and still appear in quite good condition. In both of the above forms of the disease diarrhea is variable. Diarrhea is most likely caused by a secondary proliferation of enteric bacteria. African swine fever infected pigs do not show any sign of encephalitis.

Experimentally infected pigs have had a higher than expected mortality from gastric ulcers. Fatal gastric hemorrhage most likely results from the thrombocytopenia that develops in acute ASF infection.

Laboratory Diagnosis

Laboratory confirmation is required for a diagnosis of ASF. When ASF is suspected, the Federal Area Veterinarian in Charge must be contacted and arrangements made for the submission of specimens to the Foreign Animal Disease Diagnostic Laboratory (FADDL), at Greenport, NY.

In the laboratory, specimens can be tested for ASF viral antigen, virus, and antibody. When specimens are examined for both ASF antigen and antibody, nearly all of the positive cases will be readily identified. Inoculation of porcine buffy coat cultures is a good way to isolate the virus. However, all cultures negative for hemadsorption should be tested by the direct immunofluorescent test to rule out a nonhemadsorbing ASF virus.

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The most sensitive method for isolating ASF virus is inoculation of a susceptible pig.

When performing a necropsy on a pig, remember to suspect ASF if one or more of the following are found: enlarged spleen, enlarged hemorrhagic gastrohepatic lymph node, enlarged hemorrhagic renal lymph node, and an unusual lobular pneumonia. (Dr. Charles A. Mebus, Plum Island Animal Disease Center, Agricultural Research Service, USDA, Greenport, NY 11944. 516 232-2500)

Questions about the FAD Report may be sent to:

Dr. E. I. Pilchard
APHIS, USDA
Veterinary Services
Room 801, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

Thirty days before moving, send address change, and if possible, mailing label from latest issue to:

Information Management Branch
APHIS, USDA
Room G-187, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

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Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services
Emergency Programs

Number 14-3 Fall 1986

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Current Events

Avian Influenza, 1986

On January 8, 1986, avian influenza (AI) virus (H5N2) was isolated from a chicken flock in Snyder County, Pennsylvania. During the next 12 weeks, 13 additional Pennsylvania flocks were found infected, as were four commercial operations in New Jersey, one in Massachusetts, and one in New York. These infections apparently resulted from either direct or indirect contact with live poultry markets in New York City and New Jersey. On February 1, H5N2 virus was first isolated from a live bird market in New York City. By early March, the virus had been isolated from 26 live bird markets in New York City, 12 in New Jersey, and 3 in New England. These isolations came in the wake of a survey that was initiated by Veterinary Services (VS) in the northeastern States on February 18, and completed on March 5, 1986.

Objectives of the survey were to: (1) Determine the extent of the live bird marketing system in the VS Northern Region, with emphasis on the New York City metropolitan area, (2) determine the value of live bird marketing as an AI surveillance tool, and (3) determine how the live bird industry may be operated to minimize the risk of maintaining and disseminating avian influenza virus.

It was learned that poultry, which includes ducks, turkeys, guinea fowl, chickens, and geese, change ownership 2 to 5 times between the first buyer and the consumer, when these birds are destined for live bird markets in the northeastern States. Components of the live bird marketing system include: producers—commercial integrators, backyard hobbyists, duck farms, etc.; auction markets and concentration points, dealers, wholesalers in New York and New Jersey, and retailers.

Poultry crates, trucks, and other equipment were moving avian influenza virus back and forth between producers and retailers until control procedures were initiated with industry cooperation in early February. The procedures were designed to ensure that

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the cycle of infection that was moving H5N2 virus through the marketing system (i.e., from producer to market and vice versa) was broken. Actions taken included the cleaning and disinfecting of crates, trucks, and other equipment, and the depopulation of infected poultry.

Both sodium hypochlorite solutions and One-Stroke Environ were used as disinfectants. One-Stroke was considered superior to sodium hypochlorite for the situations encountered. (See article on Approved Disinfectants, in this issue.)

New Jersey, Rhode Island, and Connecticut eliminated H5N2 virus from their few contaminated markets by depopulation, followed by cleaning and disinfection.

Initially 26 of 41 New York City markets were found to be contaminated with H5N2 virus. Most market operators were reluctant to remain free of birds for longer than 24 hours at a time. By following a regimen of 24-hour depopulations, followed by cleaning and disinfection, all markets eventually were cleaned up. Then during the week of June 23, all markets were again sampled. Four were again found to be contaminated with H5N2 virus. Evidence suggested that the latest infection reflected new introductions of the virus and that the virus had not persisted within the components of the marketing system beyond the dealers who were delivering poultry to the markets.

A survey of live bird marketing in the VS Southeastern Region was completed April 3, 1986, and extensive live bird marketing was found in Miami, Florida. Sales of live birds in the Miami area are made from "botanicas," rather than traditional live bird markets. Botanicas sell voodoo paraphernalia, including all varieties of birds for sacrificial purposes. They purchase their chickens, guinea fowl, turkeys, quail, pheasants, pigeons, and exotic birds from dealers, as well as from each other. An estimated 50 botanicas maintain birds on their premises. Birds are also held on farms, some of which are operated by botanica owners. Poultry dealers purchase chickens directly from commercial producers or, more often, from other dealers in Georgia and other southeastern States. Poultry sampled at 8 out of a total of 18 dealers and botanicas inspected in the Miami area were found to be contaminated with H5N2 virus.

Similar poultry marketing surveys conducted in the VS Central and Western Regions did not disclose the presence of H5N2 AI virus.

None of the H5N2 viruses isolated during 1986 have met the criteria established for highly pathogenic avian influenza virus (see 12-2). Indeed there have been no isolations of highly pathogenic H5N2 virus in the United States since the spring of 1984. All isolates in the VS Northern Region and Florida this year have been characterized as nonpathogenic or low pathogenic by the National Veterinary Services Laboratories. Veterinary Services' involvement in the 1986 H5N2 incidents has been strictly limited to the provision of diagnostic, epidemiological, and technical assistance to the affected States.

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Further definition of Federal, State, and industry participation in avian influenza control and eradication is expected following the International Symposium on Avian Influenza, to be held in Athens, Georgia, September 3-5, 1986. (Dr. Robert R. Ormiston, 301 436-8065)

World Animal Disease Roundup

Foot-and-Mouth Disease (FMD). A resurgence of outbreaks caused by type A_5 and one outbreak of type O FMD have been recently reported in Italy. Type A_5 had been responsible for many outbreaks dating from late November 1984 through the summer of 1985. Since November 1985, type C has been found responsible for the majority of outbreaks affecting swine. With three types of FMD virus to contend with, the situation is quite serious.

Foot-and-mouth disease in cattle was reported in Spain on June 16, 1986. Of 42 cattle on the affected farm, three had clinical signs of FMD. Proper sanitary measures were applied. No evidence of spread has been found. The source of the outbreak is unknown. The virus has been identified as A_5. Ten outbreaks of type A_5 FMD were reported in cattle and goats during the period January-August 1983.

African swine fever (ASF). Since the initial outbreak of ASF in the Netherlands on March 30, 1986, and another on April 1, 198', no further outbreaks have been reported. Clinical signs and postmortem lesions were consistent with the subacute form of the disease. The mortality rate was reported to be approximately 19 percent. A total of 6,641 swine has been destroyed, of which 196 originated from the two positive premises. Illegal feeding of uncooked garbage appears to have been reponsible for the introduction of the virus.

Four outbreaks of ASF have been confirmed in Namibia, involving more than 1,300 swine. All swine either died or were destroyed. The disease was previously reported in Namibia in October 1982. During March 1986, an outbreak of ASF was reported along the Zambia-Malawi border.

African swine fever continues to be reported in Italy (Sardinia), Spain, and Portugal.

ASF appeared last year for the first time in the Netherlands and Belgium. This points out the importance of constant surveillance for all countries in preventing the introduction and spread of ASF.

Hog cholera (HC). Since the outbreak of HC in Great Britain on April 10, 1986, nine other occurrences have been reported. Most of these were traced to a livestock market in the Midlands of England that received infected animals from a primary source outbreak. The latest diagnosis was confirmed on June 25, 1986, in an area distinct from the previous locations and not traceable to livestock markets. A total of 7,675 swine has been slaughtered in efforts to eliminate the disease. Great Britain had been free of HC for 15 years prior to the current epizootic. Feeding swine contaminated household scraps appears to be the cause of the outbreaks.

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Rinderpest (RP). Cases of RP were reported in Saudi Arabia among cattle imported from Turkey via Iraq. The animals reportedly were vaccinated in Turkey approximately 4 months earlier and revaccinated upon arrival in Saudi Arabia.

Three outbreaks of RP were reported in Uganda and one in Burkina Faso (formerly Upper Volta).

Contagious bovine pleuropneumonia (CBP) was recently reported responsible for the deaths of several hundred cattle in Nigeria. Reports of the disease continue to come from Portugal and Namibia. Based upon serological surveys and regular abbatoir inspections of carcasses, it now appears that CBP is no longer being encountered in France and Spain. (Dr. James T. Cavanaugh, 301 436-8285)

Caliciviral Disease Update

This report updates information in the September 1983 Foreign Animal Disease Report (see II-3). The 1983 information is still current, and although outbreaks of vesicular disease in domestic livestock due to calicivirus have not been recognized or reported, new and pertinent information needs to be added to the 1983 document.

Tillamook calicivirus (TCV) was first isolated from three dairy calves in Tillamook County, Oregon, in the fall of 1981. This proved to be a new serotype of calicivirus that persistently infected calves and caused vesicular lesions in inoculated swine under experimental conditions. These lesions were similar to the vesicular lesions of foot-and-mouth disease, vesicular stomatitis, swine vesicular disease, and vesicular exanthema. Neutralizing antibodies to TCV were found in California sea lion sera collected in 1983 and 1984, and in Stellar sea lion sera collected in 1976 and 1985. The antibody prevalence for these two species was 10/228, or 4.4 percent. Antibody titers using a serum neutralization test (positive at 100 percent endpoint with 100 tissue culture infective doses of virus and a lowest serum dilution of 1:20) ranged from 1:20 to 1:80. Seropositive seals were widely dispersed along the Pacific Coast from Southern California to the Bering Sea. Northern fur seals, walrus, phocid seals, and cetaceans tested negative from these same general geographic locations.

Serum samples randomly collected from cattle in the Pacific northwest were tested for antibodies against a number of caliciviruses of known ocean origin. Approximately 2 percent of the total serums tested reacted to two of these viruses. Sera were screened at 1:20 final dilution using 100 percent endpoints (4 wells in a microtiter system) and 100 $TCID_{50}$ (100 tissue culture 50 percent infectivity endpoint dose) of virus. These findings suggest that the cattle had exposure to ocean sources of calicivirus.

Experiments were then designed to examine the effect of two virus types in 4-month-old calves. The first, San Miguel sea lion virus type 5 (SMSV-5), did not produce overt disease. However the second, SMSV-13, caused vesicles at sites of intradermal inoculation on the nose and tongue. Secondary vesicular lesions

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appeared within 96 hours on the feet (interdigital). A single uninoculated pen contact calf also developed vesicular lesions on the feet. The virus was isolated from the clear fluid aspirated from these lesions. Three pen contact swine remained clinically normal.

These studies were repeated by researchers at the U.S. Department of Agriculture Plum Island Animal Disease Center using cattle about 18 months of age. Pen contact cattle remained normal. Pen contact swine developed clinical vesicular lesions.

A total of 31 calicivirus serotypes have been identified, excluding the feline and canine serotypes: 13 vesicular exanthema of swine virus (VESV) types, 12 SMSV types, and 6 other serotypes including the Tillamook virus. In 1984 the newest of the serotypes was isolated from large vesicles on California sea lions. This agent is the SMSV-13 reported above. Under experimental conditions, it is far more virulent in swine than most of the 13 serotypes of VESV originally isolated from swine.

Serological surveys of marine mammals show that SMSV-13 has been present for at least 11 years. During the 14 years that sea lions have been intensively studied, SMSV-13 was not seen to cause severe vesicular disease until 1984. Our observation is that SMSV-13 carries a virulence factor for mammals that was first expressed in 1984.

For several years, Stellar sea lions have been known to carry antibodies against certain calicivirus types. Calicivirus SMSV-6 was recently isolated from a Stellar sea lion pup on the Oregon coast by scientists at the Oregon State University College of Veterinary Medicine. Stellar sea lions range from southern California north into the Bering Sea. The virus was first isolated from a California sea lion in 1976 and from fish in 1977.

The 1983 report on caliciviral disease presented five reasons for believing that the caliciviruses of ocean origin have a potential to cause disease in man, even though no such disease had been confirmed. Then in December of 1985, a researcher at Oregon State University developed over 30 blisters on the palms of his hands and the soles of his feet. These lesions appeared concurrently with flu-like symptoms at a time when flu was quite active in the community. Direct electron microscopy of vesicular fluid aspirated from the lesions disclosed the presence of particles identical to calicivirus. The virus was isolated from the fluids and typed as SMSV-5. This serotype was originally isolated from a Northern fur seal taken in the Bering Sea in 1973. The infected researcher seroconverted to SMSV-5; however, secondary contacts (family) and others did not. There had been no known exposure to the virus, although it had been used in the laboratory. The blisters, which ranged in size from 2 to 5 mm, were quite deep and painful but were completely healed within 14 days. No additional health problems have been attributed to this virus up to the time this report was prepared, 6 months after infection. (Dr. A. W. Smith, Oregon State University, College of Veterinary Medicine, 503 754-2318)

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Avian Imports Studied

According to a recent APHIS study, the risk of an introduction of exotic Newcastle disease into the U.S. poultry industry by commercially imported exotic birds has been reduced to an acceptable level by Animal and Plant Health Inspection Service (APHIS) quarantine procedures. The study also showed that while dropping current quarantine procedures for individually imported pet birds would increase risk only slightly, some regulation is necessary to keep the risk of infected birds from entering the country at an acceptable level. The risk is particularly acute with small volume importers and smugglers. The study concluded that smuggled birds constitute the biggest risk of introducing exotic Newcastle disease into the U.S. poultry industry.

The study established an analytical framework for assessing the risk posed by imported exotic birds potentially infected with velogenic viscerotropic Newcastle disease virus. Effects of the current quarantine system on this risk were then quantified, and the change in risk that would result if the system were terminated was estimated.

Details of the study may be obtained by writing to the Policy Analysis and Program Evaluation Staff, APHIS, USDA, Room 234, 6505 Belcrest Road, Hyattsville, MD 20782.

Approved Disinfectants

Veterinary Services maintains the following list of disinfectants that have been approved by the U.S. Department of Agriculture (USDA) for use in its animal disease control and eradication programs. New disinfectants are tested and approved from time to time and are then added to the list by the Emergency Field Operations Staff, Hyattsville, Maryland.

Approved disinfectants are announced in the Federal Register.

Sodium carbonate with sodium silicate is the only disinfectant approved for use on aircraft.

Additional information on approved disinfectants may be obtained from the Emergency Field Operations Staff, Domestic Programs, Veterinary Services, 6505 Belcrest Road, Hyattsville, MD 20782, telephone area code (301) 436-8087.

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Name of Disinfectant	Percent [1]	Directions for Metric Measurement	Directions for English Measurement	Disease
Cresylic disinfectant with USDA approved mark on container	4	160 mL in 4 L of water	5.1 oz in 1 gal of water	Brucellosis Hog cholera (HC) Shipping fever Swine erysipelas Tuberculosis (TB)
Sodium Carbonate (soda ash)	-	450 g in 11 L of water	1 lb in 3 gal of water	Foot-and-mouth disease (FMD) Vesicular exanthema (VE)
Sodium Hydroxide (Lye: NaOH)	2	383 g in 19 L of water	13 1/2 oz in 5 gal of water	FMD VE
Sodium Hydroxide (Lye: NaOH)	5	1 kg in 38 L of water	5 (13 1/2 oz) cans in 10 gal of water	Anthrax [2] Blackleg [2]
Sodium ortho-phenylphenate	1	450 g in 45.4 L of water	1 lb in 12 gal of water, 60°F (15.6°C) or over	TB Infectious laryngotracheitis
One Stroke Environ [3]	0.4	1:256	1:256	HC Velogenic viscerotropic Newcastle disease (VVND)
	1	1:100	1:100	African swine fever (ASF) [4]

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Name of Disinfectant	Percent[1]	Directions for Metric Measurement	Directions for English Measurement	Disease
Sodium Carbonate[5]	4	450 g in 11 L of water with 0.1% sodium silicate added	1 lb in 3 gal of water with 0.1% sodium silicate added	FMD
Bioguard Control[3]	1	1:100	1:100	VVND
Vanodin FAM[3]	1	1:100	1:100	ASF and VVND
	0.5	1:200	1:200	FMD and swine vesicular disease

[1] Percent active ingredient in water solution

[2] Let solution remain on vehicle 8 hours and then wash away

[3] Solutions are prepared as directed on container label

[4] 60 minutes required to inactivate ASF virus

[5] Aircraft disinfectant

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FAD Report Editorial Committee

Editorial Committee membership has been changed. The current members are: Dr. E. I. Pilchard, Chairman; Dr. M. P. Dulin, Dr. R. R. Ormiston, Dr. L. J. King, Dr. M. J. Gilsdorf, Dr. J. T. Cavanaugh, Ms. Anita McGrady, and Ms. Betsy Moriarty.

Questions about the FAD Report may be sent to:

Dr. E. I. Pilchard
APHIS, USDA
Veterinary Services
Room 801, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

Thirty days before moving, send address change and, if possible, mailing label from latest issue to:

Information Management Branch
APHIS, USDA
Room G-187, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

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Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services
Emergency Programs

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Current Events

Emergency Disease Investigations, 1986

During fiscal year 1986 (Oct. 1, 1985, through Sept. 30, 1986), 165 foreign animal disease (FAD) diagnostic investigations were conducted in the United States and Puerto Rico, including 115 investigations of suspected vesicular conditions.

The first isolation of New Jersey type vesicular stomatitis (NJVS) virus this year was made at the Veterinary Services (VS) Foreign Animal Disease Diagnostic Laboratory (FADDL) in Plum Island, N.Y., from a Montezuma County, Colorado, bovine tissue submission collected July 18, 1986. The following table summarizes the results of laboratory tests of suspected vesicular diseases during fiscal year 1986:

State	Total Number of Specimens Tested	
	NJVS Virus Isolated	NJVS Antibodies Identified
Arizona	0	1
Colorado	6	27
New Mexico	0	15
Utah	2	0

Fifteen investigations of suspected hog cholera (HC) or suspected African swine fever (ASF) were completed in response to reports of either suspicious clinical signs or suspicious reactions to laboratory tests of serums collected during routine surveillance of slaughtering establishments. All investigations were HC/ASF negative. About 20,000 swine blood samples from pigs slaughtered in

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Massachusetts, New Hampshire, New Jersey, Texas, and Puerto Rico were tested at the National Veterinary Services Laboratories (NVSL) in Ames, Iowa. This testing regularly supplements our primary defenses against possible incursions of African swine fever or hog cholera.

This past summer, public health researchers working on Acquired Immune Deficiency Syndrome in Belle Glade, Fla., excited the news media when they sampled sick swine in the area and reported evidence of African swine fever (ASF). As soon as the Animal and Plant Health Inspection Service (APHIS) became aware of these reports, the pigs were quarantined under State authority. All adult hogs on three farms were examined and samples were sent to FADDL. Exhaustive testing of 126 serum samples, 43 tonsil biopsies, and other tissue specimens proved that neither ASF virus nor antibody was present. Specimens carried to Pirbright, England, by the researchers were also tested and found to be free of ASF virus.

Four VS veterinarians were sent to Mexico to gain firsthand experience in the diagnosis of hog cholera as part of ongoing efforts to maintain a cadre of experienced hog cholera diagnosticians.

On Nov. 13, 1985, velogenic viscerotropic Newcastle disease (VVND) virus was isolated from a yellow nape Amazon parrot in the State of Washington. The virus was also isolated from pet birds on the premises of the dealer from which the parrot was purchased in California. All contaminated birds were depopulated.

On March 31, 1986, VVND virus was isolated from a yellow nape parrot located at Belvidere, N.J. The infected bird had been purchased March 15, 1986, from a Cinnaminson, N.J., pet bird dealer. The dealer's 2,414 birds were depopulated on April 2, 1986, to prevent further spread of the virus. Over 300 shipments of pet birds from the Cinnaminson dealer were traced to 12 States. An additional infected dealer's premises was identified at Toms River, N.J. Three additional cases of VVND in yellow napes were reported in California during March and April. The origin of the infection in these outbreaks was not found; however, circumstantial evidence suggest that smuggled birds from Mexico were responsible.

Two VVND virus-infected, smuggled cockatoos were intercepted at the St. Thomas, U.S. Virgin Islands, airport. Both were seized and both died of VVND soon thereafter.

No VVND was identified in commercial poultry this year and the disease was again successfully excluded from pet bird trade channels.

During fiscal year 1986, a national survey for

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Nematodirus battus in sheep flocks was conducted. Thirty three thousand and six hundred one (33,601) fecal specimens from 49 States, the U.S. Virgin Islands, and Puerto Rico were examined at NVSL. Sheep were found to be affected on 33 premises in Oregon, Washington, Maryland, Vermont, and New York.
(Dr. A. E. Hall, 301-436-8073)

READI System Revised

The VS Recorded Emergency Animal Disease Information (READI) computer system has been extensively revised for user-friendly data entry and retrieval at any telephone-accessible location. The changes will permit more rapid and convenient management of data during routine investigations of suspected foreign animal diseases, as well as during a foreign animal disease task force operation. (Dr. E. I. Pilchard, 301-436-5959)

Vesicular Stomatitis Field Studies, 1982-1985

Many aspects of the biology and epidemiology of vesicular stomatitis (VS) remain a mystery despite years of study and speculation (see review in 10-3). VS is caused by two antigenically and epidemiologically distinct viruses: VS Indiana (VSI) and VS New Jersey (VSNJ). Both appear annually in tropical and subtropical areas of the Americas, whereas in the midwestern and western United States infrequent epizootic waves appear at intervals of up to 10 years. VSNJ has been the predominant serotype in epizootic vesicular disease in the United States for the last 20 years.

The disease appears to spread up and down valleys and river drainages in wet pastures, suggesting insect transmission. The main reasons for suspecting arthropod transmission are: (1) outbreaks occur in the summer at the height of the insect season, (2) VS viruses replicate in and can be transmitted by arthropods, and (3) VS virus has occasionally been isolated from field-collected arthropods. Despite these observations, doubt remains regarding the role of arthropods in natural transmission cycles. Virus can seldom be isolated from the blood of an infected animal, and low-titered viremias insufficient to infect biting arthropods are the rule in experimental studies. During the 1982-83 epizootic, 38 VSNJ virus isolations were made from biting and nonbiting flies. The former included four isolations from Culicoides midges and two from Simulium blackflies. Isolation from nonbiting flies included 27 from muscoid flies (Musca domestica and M. autumnalis), 4 from Anthomyidae, and 1 from Hippelates eye gnats. These insects were collected on premises with active clinical cases. Certainly, arthropods could play a role in mechanical transmission during an epizootic because infected cows and horses may have large quantities of virus in saliva or on surfaces of affected mucous membranes to which arthropods are attracted. Simulium and Culicoides may feed on blood or secretions and the nonbiting flies on secretions around the mouths of livestock and then transfer contaminated saliva or vesicular fluid to susceptible animals. We

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observed many flies transported in the cab of milk trucks going from farm to farm. Use of air conditioning and closed windows might reduce this risk.

It appears to be generally true that pastured animals are more frequently affected by the disease than animals in stables, barns, or dry field lots (Jonkers, A. H., Am. J. Epidemiol., 86: 288-291, 1967). A study conducted on a horse ranch in Loveland, Colo., during the 1982 outbreak showed disease in horses in a pasture that spread to animals in other pastures, and finally affected horses in barns and corrals. The incidence of inapparent infection was higher among animals in barns than among pastured animals. These findings suggest the occurrence of an environmental risk factor underlying disease expression, such as exposure to rough fodder causing mucosal abrasions. Thistles and sharp grasses could predispose to minute wounds and abrasions and allow for virus entry on contact with a contaminated animal, object, or arthropod. The progressive circumferential and centrifugal movement of virus on this farm was consistent with transmission by arthropods, although other means cannot be ruled out (Webb, P. A., et al., Am. J. Trop. Med. Hyg., in press, 1986). Certain arthropods, including Similium, Culicoides, and some mosquitoes, would be expected to feed much more readily on animals in pastures than in barns.

Contact transmission of virus between domestic animals was documented in 1982. Spread by cows dispersed from a dairy sale in Colorado and, subsequently, from heifers imported to California from Idaho. The view that outbreaks in the Western United States occur due to introduction of the virus from areas of enzootic activity, particularly the bordering Mexican States of Sonora and Chiapas, has been supported by RNA oligonucleotide fingerprinting of VSNJ strains from the United States and Mexico in 1982 and 1985 (Nichol, S. T., J. Virol., in press, 1986). Nichol found that VSNJ strains from Colorado, Idaho, Wyoming, California, New Mexico, and Nebraska in 1982-83 were identical to or very closely related to each other and to a 1982 strain from Veracruz, Mexico, but distant from the historical Ogden and Hazelhurst strains and from strains from the Southeastern United States (Ossabaw enzootic focus). Two 1985 strains from Sonora and Chihuahua, Mexico, were shown to be identical to six isolates from Arizona, New Mexico, and Colorado (from cattle and horses) but distant from the 1982-83 strains. This appears to point to known enzootic areas of VS activity in Mexico as the source of the 1982 and 1985 epizootic strains.

Serological evaluation of animals on infected premises requires tests to determine recent infection. High neutralizing (N) antibody titers persist for at least 4 to 5 years, particularly in equines, in titers up to

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1:163,840 (Webb, P. A., et al., Am. J. Trop. Med. Hyg., in press, 1986). The complement fixation (CF) test or IgM antibody capture enzyme-linked immunosorbent assay are both methods in current use to determine if infection has occurred within the past 2 months. Although N antibody can also be detected in milk samples, titers are considerably lower than in serum samples; however, milk samples are easily obtained without interrupted milking schedules. The milk testing method may provide valuable seroepidemiological evidence of past exposure to VS viruses.

Naturally occurring antibody in wildlife has been observed frequently. Antibodies were found in elk bled at high elevations in the Rocky Mountain National Park, Colorado, during 2 years preceding the 1982 epizootic. Seroprevalence rates increased between 1980 and 1981, suggesting "amplification" of virus prior to the 1982 epizootic season (Webb, P. A., et al., J. Wildl. Dis., in press, 1986). This finding suggests the virus has been repeatedly introduced or that there is a local virus reservoir. Pronghorn antelope and mule deer have high seroprevalence rates and frequently graze with domestic animals.

Studies on vaccinated herds have been limited, and the possibility that vaccine-induced N antibody is protective needs further study. Using a formalin-killed VSNJ vaccine, animals within a Colorado State University dairy herd attained a very low titered N antibody response, with peak titers (160-640) occurring about 7 weeks after inoculation and falling sharply (to 40) by 12 weeks (Gearhardt, M. A., et al., J. Am. Vet. Med. Assoc., in press, 1986). In a comparison of three dairy herds in the Pueblo, Colo., area in 1985, overt oral lesions occurred in 95 percent of one herd and peak N antibody titers reached 40,960, whereas in a dairy with only 25 percent of the herd infected (with mostly teat lesions), titers were 640. The third dairy had vaccinated their herd during the 3 preceding years; overt disease was minimal, and peak antibody titers were similar (640).

In future outbreaks, whether of epizootic proportions or limited to a few premises, field investigations need to focus on certain issues. Is the disease really irregularly distributed, skipping some herds while affecting others? It may be that overt disease expression is limited, whereas serological evidence is widespread. This must be substantiated by tests for recent infection.

If the pasture is a main disease-producing unit, intensive ecological studies may help us decide why.

Epidemiological enquiries regarding movement of animals, introduction of new animals, cograzing with wildlife, and

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recent stress factors need to be examined each time disease appears. It is particularly important to ascertain the exact number of animals on each premise, their vaccine status, and serological status.

Many questions need careful notation. Some interesting suggestions made by Dr. A. H. Jonkers are these: In herds where teat lesions predominate, is this related only to milking techniques or to cows lying in the pasture? Are the teat lesions in juxtaposition (left front, right rear), which would be expected if the animal picks up virus by lying down on it? Do cows lie predominantly on one side? Cows and horses eat differently; horses gather grasses with the lips and bite it off, and cattle wrap the tongue around the grass and tear it off. Does the site of the lesions reflect this? On farms that experience reinfection, are the same animals reinfected? (Dr. T. P. Monath and Dr. P. A. Webb, U.S. Department of Health and Human Services, Division of Vector-borne Viral Diseases, P.O. Box 2087, Ft. Collins, Colorado 80522-2087, 303-221-6404)

Zoological Animal Regulation Proposed

During recent years, increased trade in zoological animals, and hence the increase in interstate movement, has magnified the potential for disease dissemination. Past experiences with tuberculosis in bison and elk, exotic Newcastle disease in pet birds, lethal avian influenza in fancy poultry and game birds, and foreign ticks on imported rhinoceroses have caused livestock and poultry health officials considerable anxiety. Since many zoological animals are exotic to the United States, their commercial trade has substantial foreign animal disease implications.

Last year the United States Animal Health Association (USAHA) formally requested that the Animal and Plant Health Inspection Service (APHIS), United States Department of Agriculture (USDA), consider the development of a model State regulation for control of zoological animals. Through a cooperative agreement, APHIS enlisted the help of the Southeastern Cooperative Wildlife Disease Study (SCWDS), College of Veterinary Medicine, University of Georgia, to evaluate the current situation and draft a model State regulation.

Zoological animals can be broadly defined as recently captured native and non-native (exotic) wild animals, native and non-native wild animals raised in captivity, and wild forms of domesticated species. Conventional domesticated animals and some laboratory-adapted strains of wild animals are not included in this definition.

Existing regulations concerning the ownership, sale, and transportation of zoological animals are generally inadequate and are fragmented among 50 State departments of agriculture, 50 State departments of natural

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resources, the USDA, and the U.S. Department of the Interior.

A draft copy of a model State regulation was prepared that addresses ownership, sale, transportation, release, disease prevention, public safety, environmental protection, and animal welfare. Copies were submitted to Veterinary Services, APHIS, USDA, for review. At that time, APHIS officials requested that the model regulation also be made available for review by interested committees of the USAHA. These committees were asked to submit copies of the draft regulation to other organizations, particularly the International Association of Fish and Wildlife Agencies (IAFWA) and the National Association of State Public Health Veterinarians (NASPHV). Other interested organizations and individuals may request a copy of the model regulation by writing to SCWDS, University of Georgia, Athens, GA 30602. Comments and suggestions will be considered in the preparation of a revised draft, to be completed by early 1987. It is anticipated that the revised draft model State regulation will be offered to the USAHA, IAFWA, and NASPHV for endorsement. An endorsed model State regulation would then be distributed to State departments of natural resources, agriculture, and public health for their use. (Dr. V. F. Nettles and Mr. P. K. Swiderek, 404-542-1741).

Foreign Animal Disease Update

From November 26, 1984, through August 1985, 142 outbreaks of foot-and-mouth disease (FMD) serotype A_5 were reported in cattle and swine throughout Italy. In November 1985, serotype C FMD virus was diagnosed. A total of 68 outbreaks were recorded throughout the country by April 1986, mostly in swine. In June 1986, serotype A FMD virus was again diagnosed in Italy and an outbreak of serotype O FMD virus was reported. Type O had last been reported in Italy at Palermo in 1979. Since June 1986, additional outbreaks of type A FMD have been recorded in the northwest, northeast, and central regions of the country. More swine have been affected than cattle.

As a result of the Italian situation, the European Community (EC) Veterinary Committee held a meeting in Brussels, Belgium, on Sept. 2, 1986. Highlights of this meeting were reported as follows: The EC imposed a 3-month ban on Italian meat from all Italian counties where cases of FMD have been confirmed. If FMD is not controlled within this period, the ban will continue. The ban will be imposed on other counties where new FMD outbreaks are confirmed. Areas in Italy where FMD has not been confirmed can export only deboned meat, canned meat, and cured meat (that is, Parma ham) to the EC. Exports of livestock from Italy to the other member States of the EC are banned indefinitely. In addition, Yugoslavia has banned the importation of all Italian livestock and meat.

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No outbreaks of FMD have been reported in Spain since the June 16, 1986, outbreak of subtype A_5 in that country. All markets have been reopened and it appears that the situation is under control.

Malaysia reported FMD serotype $Asia_1$ in wild fauna following outbreaks in domestic livestock that began in June 1985. The Pirbright World Reference Laboratory for FMD has confirmed serotype O in samples received from Hong Kong, Nepal, Saudi Arabia, Kuwait, and South and North Yemen.

Tanzania reported occurrence of FMD serotypes SAT2 and O. Cameroon reported serotype A. Samples from Burundi contained serotype O.

In South America, FMD serotypes A and C were reported from Ecuador, serotype C from Uruguay, and serotype O from Paraguay.

Hog cholera (HC) has not been reported in Great Britain since it was last confirmed on June 25, 1986. The virus was identified on a total of 10 premises and animals on an additional six premises were serologically positive. All the swine were depopulated. It now appears that bacon that had originated on the continent and that was subsequently fed as uncooked garbage was responsible for the primary outbreaks. The source of the bacon could have been any one of three countries: Holland, Belgium, or West Germany. British veterinary officials are confident that they have established the source of the problem and that the disease has been eradicated.

There has been an upsurge of hog cholera in Belgium, with 17 occurrences since September, bringing the total to 67 since the beginning of 1986. Austria, France, East and West Germany, and Italy continue to report HC. The disease was also reported in Malaysia (Sabah) and Taiwan.

In Africa, Togo has reported three outbreaks of **Rinderpest.** In one, 6 animals died and 10 were slaughtered. Control measures were reportedly taken in all cases. Rinderpest was also reported in Egypt.

The United States is considering Belgium's request for recognition as being free of **African swine fever (ASF).** The last report of ASF in Belgium was May 2, 1985. In the Netherlands, no outbreaks of ASF have been reported since April 1, 1986 (see 14-3). Italy (Sardinia) and Portugal continue to report ASF. (Dr. James T. Cavanaugh, 301-436-8285)

New Cattle Disease in Japan

Enzootic congenital abnormalities of unknown origin have been reported in cattle in southern Japan since November 1985. Abnormal newborn calves were reported in seven

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prefectures of Kyushu Island, mainly in the southern part.

Reported Cases of Enzootic Congenital Abnormalities of Cattle by Prefecture and Month, 1986

Prefectures	Nov	Dec	Jan	Feb	Mar	Apr	May	Total
Fukuoka			1	4	8	3	1	17
Saga			1	2	1		1	5
Nagasaki			1	5	11	10		27
Kumamoto	2	7	24	32	44	6		115
Oita			27	56	67	21	8	179
Miyazaki			62	101	59	3	1	226
Kagoshima	54	274	650	517	149	34	26	1,704
TOTAL	56	281	766	717	339	77	37	2,273

At first glance, affected newborn calves may appear normal. However, some lack the ability to suckle or they suckle slowly and are weak. Some show nervous signs. Underdeveloped cerebrums and cerebellums have been observed at necropsy. However, no arthrogryposis, typical of Akabane disease, has been observed. Although the new disease has primarily affected beef cattle, it also has been seen in some dairy breeds. Characteristic congenital abnormalities have been recognized in cattle herds previously vaccinated against Akabane disease. No relationship has been established between the new disease and the reproductive history of dams.

The British National Institute of Animal Health (NIAH), Ministry of Agriculture, Forestry and Fisheries (MAFF), has been cooperating with Japanese prefectural government institutions, especially Livestock Hygiene Service Centers, in carrying out research to identify a cause of the abnormalities. Bacterial and viral examinations have not identified an etiological agent in nervous tissues and other organs of affected calves. Perivascular cuffing was observed microscopically in the brain, suggesting that the disease may be caused by an infectious agent. Skeletal muscle atrophy has not been observed, in contrast to the muscular damage frequently associated with Akabane disease.

Serological tests were conducted on fetal serum samples collected from calves showing such abnormalities using about 30 different strains of arboviruses; however, no reactions were observed. Antibodies in precolostral serum samples of affected calves were demonstrated against viruses which had been isolated from a blood sample collected from sentinel cattle kept at the Kyushu Branch Research Station of NIAH. These viruses were also isolated from culicoides there in the previous year. The viral isolates were kept at the research station in isolation from cases of enzootic congenital abnormalities. Research is in progress to characterize

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the virus. Preliminary results have identified the isolates with a member of the orbivirus group of the Reoviridae family.

The NIAH has cooperated in nationwide surveillance to assess the disease situation. The surveillance revealed 3,243 cattle including 2,159 cases in Kyushu with congenital CNS symptoms or weakness, without other physical abnormalities, during the period between November 1985 and March 1986. Other diseases were found, including Akabane in Japan and bovine viral diarrea (BVD) in 47 calves in Hokkaido. (Excerpted from O.I.E. Asian Information No. 58, Asian Bureau of the Office International des Epizooties, Tokyo, Japan, by Dr. James T. Cavanaugh, 301-436-8682)

Screwworm Program Update

The Mexico-United States screwworm eradication program was plagued by several isolated outbeaks north of the sterile fly barrier at the Isthmus of Tehauntepec in Mexico during 1985. (See June 1985 issue, 13-2 for previous update.) Most of these were associated with animal movement from an infested area in southeast Mexico. The largest outbreak occurred in the States of Tamaulipas and San Luis Potosi where 139 confirmed screwworm cases occurred between June 25 and Aug. 8, 1985. The origin of this outbreak was not established; however, animal movements or intentional transport of fertile screwworm insects was suspected. The last two cases to be reported north of the barrier were collected from animals that had been transported from southeast Mexico to the state of San Luis Potosi in November 1985.

Inspection and quarantine stations located on the only highways crossing the Isthmus of Tehuantepec began operations prior to January 1985. All animals being transported across the Isthmus by these routes or by either of the two railroads are inspected, sprayed, or dipped in an effective larvicide, and larvicide is applied locally to any wounds. Infested animals are quarantined for 3 days while wounds are treated and observed. Quarantine enforcement is augmented by the presence of Mexican military personnel at each station.

An extensive information campaign was initiated in November 1985 for transporters and livestock owners at the points of origin of animal shipments. They were encouraged to inspect animals at points of origin and avoid transporting animals infested with screwworms. The result was a reduction in the number of infested animals presented at inspection and quarantine stations. From October 1985 to September 1986, 326,514 animals were inspected and 163 quarantined due to screwworm infestation. No screwworms have been found north of the sterile fly barrier since November 1985.

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In August 1985, the Mexico-United States Commission for the Eradication of Screwworms approved the development of an amendment to the 1972 agreement which formed the Commission. The amendment authorized the Commission to extend the program into the Yucatan Peninsula, cooperate with international organizations, and negotiate and sign agreements with countries of Central America and Panama for the eradication of screwworms. The amendment was ratified by Mexico and the United States on April 1, 1986. An eradiction program in the Yucatan Peninsula began on May 19, 1986. By September, the numbers of reported cases from that area had been reduced. Most of the cases were near the border of Belize and Guatemala.

There have been discussions with the Governments of Guatemala and Belize about a screwworm eradication program. Agreements between the Mexico-United States Commission and those countries have been drafted and final negotiations are in progress.

The construction of a new plant for the production of sterile male screwworm flies in Panama is being planned. The new plant will have a capacity of at least 200 million sterile screwworm flies per week. An agreement with Panama is being drafted. Contracts with a construction management company and an architectural and engineering company will be awarded by the end of 1986. (Dr. J. E. Novy, Mexico-U.S. Commission for the Eradication of Screwworms, Apartado Postal M-2890, 06000 Mexico D.F., Mexico, 905-250-5992)

Research at Plum Island

(Note: The following report is the first of a series on foreign animal disease research at the Plum Island Animal Disease Center (PIADC), Agricultural Research Service, U.S. Department of Agriculture, Greenport, N.Y.)

Survival of several swine viruses in Parma ham was studied to determine the safety of importing this specialty product into the United States.

The name "Prosciutto di Parma" (Parma ham) is reserved exclusively for ham having characteristic qualities related to the geographical area of its production and the various phases of preparation from salting to the end of the curing period. Preparation and curing are carried out in a designated area within the Province of Parma, which is located in the Po Valley in northern Italy. The ham is obtained from heavy adult castrated male swine reared under controlled conditions. The curing period must not be less than 12 months. The United States Government banned the importation of these hams because of outbreaks of African swine fever (ASF) and swine vesicular disease (SVD) in Italy between 1973 and 1978. Cooperative studies were undertaken at PIADC, the Instituto Zocprofillattico Sperimentale, Brescia, Italy, and the Instituto Zooprofillattico Sperimentale, Sassari, Italy, to determine if the viruses of FMD, SVD, ASF, and

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hog cholera (HC) were inactivated by the processing and curing procedures used in preparing Parma hams. Hams were prepared from pigs infected with each of these diseases when the carcass contained the greatest amount of virus. Thus, these experiments are examples of a "worst case situation," and in general hams would contain more virus than would probably occur under field conditions. SVD virus was not recovered after 360 days, FMD virus after 170 days, ASF virus after 399 days, and HC virus after 313 days. The processing and maturing of the ham inactivates these viruses and such hams, if processed through a 400-day period, they would be unlikely to be a source of disease transmission. (Dr. P. D. McKercher, PIADC, Greenport, N.Y., 516-323-2500)

Focus on... Venezuelan Equine Encephalomyelitis

Venezuelan equine encephalomyelitis (VEE) is an acute, infectious disease of equines and humans caused by an arthropod-borne virus classified in the alphavirus group (group A arbovirus) of the Togavirus family. The disease is transmitted by mosquitoes and possibly other hematophagous insects. The disease was first reported in South America, where it is known as "peste loca." Horses, mules, and donkeys are equally susceptible to VEE virus.

History

VEE virus was first isolated in 1936 by guinea pig inoculation of a brain suspension from an encephalitic horse in Venezuela. The virus was shown to be different from Western (WEE) and Eastern (EEE) equine encephalomyelitis viruses in cross-protection studies in guinea pigs. Review of the literature suggested that VEE probably has occurred in Venezuela, Colombia, and Peru since at least the early 1900's. Epizootics of VEE occurred yearly from 1936-73, with major peaks of activity at 6- to 10-year intervals. Tens of thousands of equines were clinically affected and thousands of deaths occurred during peak periods. The first VEE-related human illness was reported during a major outbreak in Trinidad in 1944. Mansonia titillans mosquitoes were incriminated as the virus vector. Outbreaks in Venezuela during the late 1950's and 1960's and laboratory infections among researchers confirmed that VEE virus was pathogenic for humans. Infection in humans produces a "flu-like" illness that has been described as a "most unpleasant illness, memorable for years." Encephalitis and deaths were most commonly reported in young children and adolescents.

The most recent VEE activity began in Venezuela and Colombia in 1969 as a devastating epizootic that spread to Ecuador and Peru. In an unprecedented occurrence, VEE virus was transported in an unknown manner to an area on the Pacific Coast of the border between Guatemala and El

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Salvador in late June 1969. An epizootic wave of VEE virus activity moved relentlessly through the equine population of all Central American countries (except Panama) and Mexico, eventually reaching the United States (Texas) in late June 1971. Approximately 142 equines in the United States were confirmed to have died from VEE during this activity in 1971. The last documented cases of VEE in North America occurred in Mexico in 1972. In South America, the last cases of VEE in equines occurred in the Goajira region of Venezuela in 1973. The 1969-1972 epizootic of VEE was modified and ultimately interrupted by the use of an attenuated VEE vaccine and aerial spraying of ultra-low volume (ULV) malathion over millions of acres of land in Texas and Mexico. Epizootic VEE activity has not been reported worldwide since 1973.

Geographic Distribution

Epizootic VEE has occurred naturally in Venezuela, Colombia, Trinidad, Peru, Ecuador, Costa Rica, Nicaragua, Honduras, El Salvador, Guatemala, Belize (formerly British Honduras), Mexico and the United States (Texas). Some epizootics in Argentina and Venezuela have been ascribed to equine vaccination with an incompletely inactivated formalin-treated VEE virus vaccine. Epizootic VEE has been associated with equines in areas of Latin America ecologically classified as tropical dry or tropical thorn forests. These areas have prominent dry seasons and are the traditional cattle-raising and rice-growing areas of the tropics. Here VEE epizootics may continue with the presence of mosquito vectors as long as susceptible equines are available. Human infections typically follow equine disease by approximately 2 weeks. Humans are tangentially infected from equines; epizootics and epidemics cease when susceptible equines are no longer available.

During the 1960's, VEE virus activity was reported in areas of tropical and subtropical America where VEE epizootics in equines had never been reported. Occasional human illnesses and fatalities confirmed the pathogenic nature of the VEE isolates; however, equine disease did not occur.

Virus Subtypes and Variants

In a classical study, Young and Johnson (Amer. J. Epidemiol. 89: 286, 1969) applied a short incubation hemagglutintion inhibition (HI) test by using immune sera produced by the inoculation of the spiny rat, Proechimys semispinosus, to test all available isolates of VEE virus. The antigenic relationships observed were stable upon passage and were not related to the donor host from which the virus was isolated. They showed that place of origin and, to a lesser extent, time were important determinants of antigenic variation, that isolates could be differentiated geographically and that certain isolates were associated with epizootics, while others were not. These researchers separated the isolates into

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four subtypes (I-IV); and with subtype I, they identified five variants (A-E). Later studies identified subtype I variant F, two variants within subtype III (A and b), and two additional subtypes (V and VI). These studies confirmed the Young and Johnson distribution, except for subtypes I-A and I-B, which were shown to be identical and are now referred to as I-AB. Young and Johnson recognized that all VEE isolates associated with equine disease were antigenically grouped as subtype I, variants, A, B, and C (epizootic variants). These variants have only been isolated during equine epizootics; isolations ceased when there was no more epizootic activity. It was presumed in 1969 that an equine epizootic with the other subtypes/variants awaited only the presence of competent vectors and susceptible equines.

Prior to 1969, variants I-AB and I-C were isolated only during equine epizootics in northern South America (Venezuela, Trinidad, Colombia, Ecuador, Peru, and Argentina). In contrast, the other subtypes and variants were demonstrated continuously over time in areas where few equines or humans ventured, tropical wet forests and shaded fresh-water swamps. Such areas included the Florida Everglades (subtype II); discontinuous foci from Veracruz, Mexico, to Almirante, Panama (variant I-E); an area from the Panama Canal Zone to Colombia and Venezuela (variant I-D); and Trinidad, Brazil, Suriname, and Guyana (subtypes III-A, III-B, and IV). While occasional outbreaks of VEE occurred in humans, equine VEE was not reported. In later studies, variant I-F was demonstrated in Brazil; subtype III-B was found in an unusual association with swallow bugs and birds in ecologically arid areas of Colorado and Utah; subtype V was isolated in French Guiana; and subtype VI was isolated in Argentina.

With the outbreak of VEE in Central America in 1969, it was initially presumed that the long-expected epizootic expression of variant I-E had occurred. The revelation that the epizootic variant was I-AB, identical to the epizootic virus concurrently active in Ecuador at that time, forced a reevaluation of VEE viruses. It is now accepted that the variants I-D, I-E, and I-F, and subtypes II, III, IV, V, and VI are sylvatic viruses found in enzootic foci. **The sylvatic VEE viruses do not cause VEE in equines, but are pathogenic for humans.** There are no data to suggest that epizootic (equine virulent) VEE variants arise from sylvatic VEE viruses in enzootic foci. While epizootic VEE and the epizootic VEE variants have not occurred since 1973, the sylvatic VEE viruses in enzootic foci currently persist in many areas of subtropical America.

Transmission

The primary means of transmission of both epizootic and sylvatic VEE viruses is by mosquitoes. Vector mosquitoes

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are found in virtually every aquatic habitat in tropical America. The sylvatic viruses circulate among rodents and birds by the feeding activity of mosquitoes primarily in the genus Culex subgenus Melanoconion. Humans and equinea seldom venture into the enzootic focus of activity.

Infection of humans with either sylvatic or epizootic VEE viruses can produce severe clinical disease, encephalitis, and death. Infection of equines with sylvatic VEE virus causes low level viremias but no clinical disease; infection does, however, confer cross protective immunity in equines to subsequent infection with epizootic VEE virus.

Due to the high concentrations of viremias in infected equines, epizootic VEE viruses can be transmitted by many species of hematophagous insects. At least 34 species of 8 genera of mosquitoes have been implicated as biologic vectors of epizootic VEE viruses. The most important epizootic VEE virus vectors are Mansonia titillans, M. indubitans, Psorophora confinnis, Ps. discolor, Aedes taeniorhynchus, A. sollicitans, A. aegypti, A. thelcter, A. scapularis, and Deinocerites pseudes. In addition, Simulium spp. and Culicoides spp. have been suggested as biologic or mechanical vectors. Epizootic VEE virus can be moved vast distances in a short period of time.

Typically for arboviruses, including VEE virus, transmission occurs with the feeding of infected female mosquitoes on susceptible hosts. The virus must replicate during an extrinsic incubation period in the biological vectors before virus can be transmitted via the salivary glands to the host.

While aerosol transmission to humans commonly occurs in laboratory-acquired infections, aerosol or direct contact transmission in naturally infected equines is negligible. Natural human infection with epizootic VEE virus occurs tangentially from the feeding of vector mosquitoes on infected equines and subsequently on humans.

Pathogenesis

Within 12 to 24 hours after infection, a febrile response and viremia occur in equines. Fever may reach 104°F (40°C) and viremia may exceed $10^{5.5}$ $SMICLD_{50}$ (suckling mouse intracerebral median lethal doses)/ml of blood.

Viral titers may occasionally exceed $10^{8.8}$ $SMICLD_{50}$/ml. At approximately 5 days after the onset of infection, viremia ends, neutralizing antibodies can be demonstrated, and initial signs of central nervous system (CNS) disease appear. Clinical signs may vary from a mild, febrile response to severe CNS disease with stimulation, hyperexcitability, hyperesthesia, profound depression, and death. Signs may also include leukopenia with profound lymphopenia, psychic depression with

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drooping ears, and a somnolent appearance, trismus, inability to swallow water, frothing at the nostrils and mouth, anorexia, walking in circles and into obstacles, and pressing of the head against obstacles. Death may occur within 5 to 6 days or as late as 9 to 14 days after the onset of infection. Animals with mild infections may recover and have no neurologic sequelae.

Lesions

In the equine, VEE causes lymphoreticular destruction and encephalitis. Visible lesions are the result of self-inflicted trauma. Lesions may include contusions and abrasions on the skin and hemorrhages of the surface of the brain and meninges. Microscopic changes include encephalomyelitis and hemorrhages of all types in the brain and meninges.

Diagnosis

A tentative diagnosis of viral encephalomyelitis in equines may be based upon the occurrence of acute neurologic disease during the season in which biting fly activity occurs and in the presence of large numbers of mosquitoes and hematophagous insects. Typically, VEE virus will cause clinical disease in many equines at the same time rather than cause individual cases. Confirmtory diagnosis during an epizootic of equine encephalomyelitis must be based upon isolation and identification of virus or upon seroconversion demonstrated with the complement fixation, hemagglutination inhibition, or neutralization test. The differential diagnosis of VEE includes eastern and western equine encephalomyelitis and other less frequently reported arbovirus-caused encephalitides, rabies, African horsesickness, equine infectious anemia, toxic encephalitis, mineral poisoning, botulism, shock, hepatoencephalopathy, protozoal and verminous encephalitis, and leukoencephalomalacia.

Due to the occurrence of sylvatic VEE virus in many parts of tropical America, a diagnosis of VEE in individual equines based upon serologic studies alone must be made with extreme caution. Sylvatic VEE virus infection in equines will produce antibodies against epizootic VEE viruses that can be differentiated only by the viral neutralization test.

Virus isolation is most easily completed by intracranially inoculating suckling mice or inoculating cell cultures with blood collected from febrile, clinically normal equines located near encephalitic equines. VEE virus is then identified by the HI or neutralization test.

Control

During the most recent epizootic in 1969-72 in North America, control was effected by a combination of vector control by the aerial application of insecticide and vaccination of equines.

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The vaccine used had been earlier developed for use in humans. The use of a human vaccine in equines was unprecedented in the annals of virology. This live, attentuated vaccine, strain TC-83, proved to be safe and effective for equines when properly applied. Millions of equines were safely vaccinated during and after the outbreak. Immunity to epizootic VEE infection occurs within 5 days after vaccination and probably persists for life.

A formalin-inactivated VEE vaccine produced from virus replicated in embryonated chicken eggs was used for decades in Latin America. Experience has shown that VEE virus is exceptionally difficult to inactivate. Numerous outbreaks of VEE have been traced to the use of such formalin-treated preparations. Formalin-inactivated VEE vaccines made with virulent or nonattenuated viruses should never be used in equines or humans.

A trivalent VEE, EEE, WEE formalin-inactivated vaccine is commercially available in the United States. The VEE virus antigen used is strain TC-83. The vaccine is safe and effective.

Summary

Clinical VEE in equines is caused only by certain variants of VEE virus. Epizootic VEE virus has not been isolated and clinical VEE has not been confirmed in equines since 1973. Sylvatic variants and subtypes of VEE virus are commonly isolated in tropical and subtropical America. Sylvatic strains of VEE virus may cause clinical encephalitis in humans, but are nonpathogenic for equines. (Dr. Thomas E. Walton, USDA, ARS, Arthropod-borne Animal Diseases Research Laboratory, P.O. Box 3965, University Station, Laramie, WY 82071)

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Questions about the FAD Report may be sent to:

Dr. E. I. Pilchard, Editor
APHIS, USDA, VS
Room 801, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

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